A multi-faceted approach towards interpreting early life experience and infant feeding practices in the ancient Atacama Desert, Northern Chile.

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Abstract

Interpreting early life experience in the past is of considerable interest to archaeologists, yet remains fraught with difficulty. Children are sensitive barometers of population health in general. In addition, infant feeding practices, and stresses experienced during infancy and childhood, have important effects on adult health and demographic changes. Understanding how diet and physiological stress interact is therefore of significance, but most bony indicators of stress have non-specific etiologies that cannot be tied to events during childhood. The recent advent of incremental isotopic techniques means we now have the potential to identify periods of stress and dietary change during childhood through changes to stable carbon and nitrogen isotopic ratios during tissue formation. Being able to establish these individual weaning trajectories allows us to consider individuality in past weaning choices, giving the bioarchaeologist a more nuanced picture of the past. Here we investigate whether combining paleopathological and isotopic data can give insight into the synergy between infant feeding and stress. We present a case-study of a five-year-old child from an early agricultural period archaeological site in the northern Atacama Desert, Chile. We show that, despite physiological stress likely affecting isotopic ratios in this individual, a weaning curve is visible and the interpretation of weaning behavior is possible. In addition, we suggest that there is isotopic evidence for a micronutrient deficient weaning diet that may be correlated with bony evidence for pathology.

Introduction

The study of early life experience, and particularly weaning practices, is central to many anthropological and archaeological questions. Infant feeding practices are related to diverse

factors including maternal and infant health, subsistence base and availability of complementary foods, childcare practices, reproductive decision-making, and cultural expectations (Larsen, 2015; Lewis, 2007; Stuart-Macadam & Dettwyler, 1995). Because of this, the study of weaning has been a focus of bioarchaeological research (see Mays et al., 2017 for a review). The complexity of the weaning process and synergistic relationship between infant diet and infant health, however, make interpreting past behavior challenging (Lewis, 2007; 2018). In this study we aim to assess whether the use of incremental isotopic analysis in combination with more traditional paleopathological techniques can enable better interpretation of the relationship between infant-feeding practices and physiological stress.

There are several issues with the bioarchaeological interpretation of infant-feeding practices and their relationship with childhood health. Chief among these is that the term 'weaning' is not applied uniformly by all bioarchaeologists. In most cases, weaning is a process which takes years to complete, rather than a single event (Katzenberg et al., 1996; Millard, 2000). This process begins when the mother begins to supplement the infant's diet with non-breastmilk foods and ends when breastmilk no longer forms a component of the diet. It is important to differentiate these different stages in the weaning process, as health will not be affected in the same way throughout the process. The infant or child is likely to experience the greatest exposure to stressors at the initiation of supplementary feeding, when the transfer of passive immunity from the mother via breastmilk decreases and the infant's own immunity is still developing (Lawrence & Lawrence, 2010; Lönnerdal, 2000; McDade & Worthman, 1998). Although continued breastfeeding during the process of weaning has immunological benefits (Labbok et al. 2004), there is compelling evidence from clinical epidemiological and immunological development research that the *start* of weaning results in the poorest outcomes in infants younger than one year of age (Gordon et al. 1964, Simon et al. 2015).

In addition, 'weaning stress' is a problematic descriptor, used by many to describe a period or periods of physiological stress experienced by the child due to weaning (Halcrow et al., 2017). As the end of the weaning process is more easily identified using bulk isotopic methods (Millard, 2000), archaeological interpretations of 'weaning stress' more often focus on this point. In this study we use the term physiological stress, rather than weaning stress. This includes any physiological response to an external stimulus that results in systemic stress (Goodman et al., 1984; Temple & Goodman, 2014). In using this term we acknowledge that many skeletal indicators of physiological stress have non-specific etiologies (Katzenberg et al., 1996), meaning that they cannot be definitively tied to the weaning process.

Skeletal and dental indicators of physiological stress which may be linked to the weaning process:

In this study we look particularly at porotic hyperostosis lesions, which are often considered to be early-life stress indicators (e.g. Lewis, 2018). Porotic hyperostosis has a range of possible etiologies but is generally associated with forms of anemia (Oxenham & Cavill, 2010; Walker et al., 2009). These lesions cannot be assigned precise formation times, instead the bioarchaeologist can only note whether or not lesions are actively forming at time of death, and

consider whether the weaning process, and particularly micronutrient deficient complementary foods, may have been responsible based upon the age of the individual (Ortner, 2003; Walker et al., 2009). Isotopic evidence can be a helpful addition here too, as it may reveal when and if nutrient deficient weaning foods were introduced to the individual, potentially resulting in the observed lesions.

Linear enamel hypoplasia (LEH) are developmental defects in enamel caused by a disruption in enamel-matrix apposition, often due to periods of nutritional inadequacy and/or infection (Hillson & Bond, 1997). They can be assigned estimated times of occurrence based on their position in the tooth (Reid & Dean, 2006). Weaning stress has been generally considered responsible for peaks in LEH formation between 2-4 years of age in a number of studied populations, primarily on the basis that this is when these groups were likely to have weaned (Blakey et al., 1994; Sellen, 2007). However, there is increasing recognition that timing of events in the weaning process (i.e. initiation of supplementary feeding and weaning completion) must be identified using other methods, such as isotopic analysis, before these hypoplasias can be definitively associated with weaning (Temple, 2016).

Isotopic identification of weaning:

Isotopic interpretation of weaning is based upon the premise that exclusive breastfeeding involves a trophic level shift, during which time the infant or child is one trophic level above maternal isotopic values (2-3‰ for $\delta^{15}N$ and around 1‰ for $\delta^{13}C$ (Fogel et al., 1989; Fuller et al., 2006; Jay et al., 2008)). As the child is weaned onto a diet reflecting that of adults in the sample their isotopic values decrease gradually, until they align with adult values (Fogel et al., 1989; Fuller et al., 2006; Jay et al., 2008). The isotopic identification of population weaning behavior has been primarily undertaken using cross-sectional isotopic sampling (Jay et al., 2008; Millard, 2000; Schurr, 1998; Tsutaya & Yoneda, 2013). Cross-sectional analysis involves sampling the bone collagen of as many infants and children as possible, and comparing their carbon and nitrogen isotopic ratios to adult dietary baseline values (see Tsutaya & Yoneda, 2015 for a review). The point at which the subadult values begin to match adult values is considered the end of weaning, and there are various statistical modeling methods that may be used to define this timing of weaning completion within the population (Millard, 2000; Tsutaya & Yoneda, 2013; 2015). To evaluate the effects of physiological stress during the weaning process, however, we need to be able to look beyond population-level interpretations of the weaning process. The timing and duration of the weaning process will differ from person to person, and they will therefore be most vulnerable to stressors at different times.

Understanding individual weaning patterns is therefore useful in assisting with the interpretation of skeletal stress markers, including those associated with the weaning process. Using dental tissues that grow at known rates, changes to diet throughout infancy and childhood can be established, to build a picture of when weaning was initiated and the point at which protein from breastmilk no longer formed a significant proportion of the diet (Beaumont et al., 2013; Beaumont & Montgomery, 2015; Burt & Garvie-Lok, 2013; Eerkens et al., 2011;

Henderson et al., 2014). In addition, these isotopic profiles may give insight into the weaning diet used, particularly if it involved different resource-types than were typical for the adult diet. Beaumont and Montgomery (2016), for instance, showed that, in a European context, a shift to a maize-based diet during childhood is clearly visible in isotopic profiles. Incremental isotopic profiles may also highlight periods of physiological stress (Fuller et al., 2005; Mekota et al., 2006; King et al., 2018a). Nitrogen isotopic ratios, for instance, have been shown to rise during periods of tissue catabolism, with correlated changes in carbon isotopic ratios (Fuller et al., 2005; Mekota et al., 2006).

There are multiple explanations for changes to isotopic values including simple dietary change, physiological stress, or some combination of both processes. When looking at the weaning period, a high nitrogen isotope value may be due to exclusive breastfeeding, nutritional stress relating to the weaning process, or physiological stress unrelated to infant-feeding practices. These processes cannot be disentangled when looking at a single datapoint, as is generally the case when using bulk bone collagen samples. However, using incremental isotopic techniques has the potential to help circumvent interpretive issues (King et al., 2018c). An incremental profile yields a series of results which means a high nitrogen value can be seen in context. If high isotopic values are the result of exclusive breastfeeding these datapoints should be followed by a series of isotopic values forming a gradual curve to lower values as the child is weaned (as described above). If physiological stress is responsible for values we might expect to see consistently high nitrogen isotope values in periods of prolonged stress, followed by sudden decreases in values as stress is reduced. Sudden spikes in nitrogen values, on the other hand, may be linked to periods of acute stress (Beaumont & Montgomery, 2016; King et al., 2018a). However, there remains a level of interpretive difficulty as exclusive breastfeeding and prolonged stress will look isotopically similar, particularly in terms of nitrogen isotope values. In these instances, it may be useful to consider other lines of evidence relating to physiological stress alongside isotopic results.

Combining skeletal and isotopic indicators:

Using paleopathological indicators in conjunction with incremental isotopic analysis may assist in unravelling interactions between nutrition, stress, and mortality in past populations (Halcrow et al., 2017). By building isotopic profiles showing when weaning is initiated, and the duration of the weaning process, we can begin to understand when/if physiological stress is occurring relative to the weaning process (King et al., 2018a). This will help us to assess whether osteological evidence can be linked to the weaning process. Conversely, by looking at osteological evidence we may be able to evaluate whether isotopic values are likely to be affected by physiological stress, allowing us to make more nuanced interpretations of results.

Here we assess whether the combination of these techniques can help to tease apart early-life stress events using a case study from the early agricultural (Formative) period of the northern Atacama Desert, Chile. This individual forms part of our wider study of infant-feeding and stress in the Atacama context and was chosen because of their bony evidence for stress during early life (King et al., 2018a). Many isotopic studies avoid sampling obviously pathological

individuals because of the potential that stress will obscure the isotopic signals of dietary change (Katzenberg & Lovell, 1999). However, the majority of infants and children in the archaeological record will have died due to ill-health, whether or not this was sufficiently chronic to manifest skeletally (Wood et al., 1992). In this study we look at an individual with obvious pathology in order to establish whether weaning trajectories are still visible isotopically when physiological stress is also affecting the individual, and whether aspects of the incremental isotopic profile might reveal when these stresses were experienced.

The harsh environment of the Atacama Desert is a useful place to examine early-life stress experiences, as environmental stressors are plentiful. Thus, there are a number of possible etiologies for stress in individuals from the Atacama, with 'weaning stress' being just one of the options. As the driest hot desert in the world, the Atacama is a marginal environment. Agricultural resources are extremely vulnerable to El Niño oscillations, and the only cultivatable areas lie in the snowmelt fed river valleys which drain the cordillera. Added to this, the rivers have naturally high concentrations of heavy metals, such as arsenic leading to potential for toxicosis in the Atacama populations (Bartkus et al., 2011; Byrne et al., 2010; Swift et al., 2015). Previous bioarchaeological research in the area has suggested that fetal and infant mortality, complications during pregnancy and childbirth (Arriaza, 2005; Arriaza et al., 1988; Arriaza et al., 2010), and micronutrient deficiencies (Snoddy et al., 2017) were common. As such, the weaning process is likely to be occurring against a backdrop of significant environmental stress.

Materials and Methods

A single individual (Az122 T9) was chosen for analysis based upon macroscopic observation of pathological conditions which may be linked to dietary deficiencies (i.e. porotic hyperostosis). Az122 T9 was analyzed using both standard osteological techniques and incremental isotopic analysis of a deciduous first molar. The information gleaned from each of these lines of enquiry was then compared to build a picture of early life experience.

The individual and their context.

T9 is from Azapa 122, one of the early agricultural sites of the Azapa Valley, close to Arica, in northern Chile (Figure 1). The site dates to the Alto Ramírez phase of the Formative Period (500BC – 200AD) (Muñoz, 2012). Archeozoological and paleobotanical analyses from Az122 and other contemporary sites indicates that subsistence in the Formative Period was based upon a variety of agricultural resources including the primary crops of quinoa (*Chenopodium*), and maize (*Zea mays*), and domesticated camelids (*Lama glama*, *Vicugna pacos)*. Other crops including tubers such as potato (*Solanum*), ullucu (*Ullucus*), and manioc (*Manihot*) are also likely to have been cultivated alongside squashes (*Curcurbita*), and legumes (e.g. *Phaseolus*) (Muñoz, 2012). Isotopic analysis of adults from this time period indicates that, although maize was available, individuals retained a broad-spectrum resource base with marine resources still a significant component of the diet (Bonilla et al., 2016; King et al., 2018b; Pestle et al., 2015; Santana-Sagredo et al., 2016). It is possible that agricultural resources comprised a more

important portion of the diet for weaning infants and children (King et al., 2018a), as crops such as maize and quinoa are easily reduced to gruels, which are used in weaning even today by the indigenous groups of the Andes (Barton et al., 2012). There is no evidence that camelids in the region have ever been used for their milk (Gade, 1999).

[Figure 1 near here]

Az122 T9 is a child who was around five years of age at the time of death. They are represented by an intact cranium only. Age was estimated using development of the maxillary dentition following AlQahtani et al. (2010).

Paleopathological analysis:

The surfaces of the cranium were examined macroscopically, and the type and distribution of abnormal bony changes were recorded. Bony lesions were defined following Ortner (2003), with any abnormal bony formation or destruction, as well as abnormalities of density, size and shape of bone being recorded. The cranium was also radiographed from the lateral and anterio-posterior view (60kV, 150mA, exposure time 150ms, at a distance of 100cm) to allow more complete description of the observed pathological lesions.

The surfaces of all available teeth were examined under both natural and artificial light, and any hypoplastic dental enamel defects were recorded using the criteria of Goodman and Rose (1990).

Incremental isotopic analysis:

The maxillary left deciduous first molar was sampled for incremental isotopic analysis. On average, this tooth forms between 0.2 years before birth and 3.5 years after birth (AlQahtani et al., 2010). Dentine was prepared for incremental analysis through mechanical removal of enamel and surface particulates using a dental drill and burr. Dentine was then partially demineralized and sliced into 1mm increments using a surgical steel scalpel (as per Beaumont et al. (2013) method 2). Collagen was extracted from each dentine increment following a modified Longin method (Longin, 1971), detailed in King et al. (2018a). Collagen was analyzed at the Stable Isotope Biogeochemistry Laboratory, Durham University. Total organic carbon, total nitrogen content and stable isotope analysis of the samples were performed using a Costech Elemental Analyzer (ECS 4010) connected to a ThermoFinnigan Delta V Advantage isotope ratio mass spectrometer. Carbon isotope ratios were corrected for ^{17}O contribution and reported in standard delta (δ) notation in per mil (‰) relative to Vienna Pee Dee Belemnite (VPDB). Isotopic accuracy was monitored through routine analyses of in-house standards, which were stringently calibrated against international standards (e.g., USGS 40, USGS 24, IAEA 600, IAEA N1, IAEA N2). Analytical uncertainty in stable carbon and nitrogen isotope analysis was typically ±0.1 ‰ for replicate analyses of the international standards and <0.2 ‰ on replicate sample analysis.

Az122 T9 was analyzed as part of our broader incremental study into weaning behavior in the Atacama (King et al., 2018a). Total organic carbon and nitrogen data was obtained as part of the isotopic analysis using an internal standard (Glutamic Acid, 40.82 % C, 9.52 % N). Collagen quality was assessed using laboratory protocols based on DeNiro (1985) and was considered to be of good quality if the C/N ratio = 2.9 – 3.6 and 35 – 50% carbon and 11 – 16% nitrogen. The time period represented by each increment was calculated according to the method developed by Beaumont and Montgomery (2015), which takes into account the individual tooth type and its formation time (AlQahtani et al., 2010).

Results

Paleopathological analysis:

Trabecular expansion (porotic hyperostosis) was observed through the ectocranial surface of the frontal, occipital, and posterior parietal bones (Figure 2). Radiographs support this diagnosis, showing clear expansion of the diplöe, particularly in the occipital (Figure 3). There is also subperiosteal new bone formation on the anterior maxilla. There is no evidence for remodeling of this new bone or the porotic hyperostosis, indicating that these lesions were active at time of death.

In addition to this evidence for pathology, the cranium is smaller than expected for an individual of five years of age, though there are no population-specific reference collections against which this can be quantified. The cranial length (glabella – opisthocranium) is 137.1 mm, maximum cranial breadth is 120 mm and cranial height (basion – bregma) is 127.6mm. The individual exhibits premature fusion of almost all cranial sutures, and patchy thinning of the cranial table associated with intracranial pressure (known as ‘copper-beaten appearance’ (Figure 3; Agrawal et al., 2007)). The sutures of the vault are all obliterated, except for the inferior part of the metopic suture (Figure 2), unusual in itself as this suture is normally closed by 9 months of age, and certainly by two years (Weinzweig et al., 2003). Many of the facial sutures are also affected, the zygo-maxillary suture is partially closed on both sides, and only the fronto-zygomatic suture remains partially open. The cranium is slightly asymmetric, with left-side occipital flattening and tilting of the cranial base (Figure 2). The nasal aperture also appears asymmetric. The overall skull-shape is dome-shaped (oxycephalic). This increase in cranial height is generally associated with early closure of the lambdoid and coronal sutures (Barnes, 1994). The premature fusion of the cranial sutures, often associated with copper-beaten appearance, is clinically known as craniosynostosis and has a number of etiologies, as discussed below. In addition, the maxilla shows retrusion, which has reduced the prognathism of the mid face.

[Figures 2 and 3 near here]

No dental enamel defects were recorded. However, only the left maxillary deciduous first and second molar are present. We acknowledge that linear hypoplasic enamel defects are unlikely

to be observed in deciduous dentition, particularly when the anterior dentition are missing, as is the case in this individual (Goodman & Armelagos, 1985).

Incremental isotopic analysis:

The incremental isotopic results for Az122 T9 are given in Table 1 and graphically in Figure 4. Expected isotopic changes during the weaning process, as detailed in the introduction, are also given for comparison in Figure 4. In Az122 T9, $\delta^{15}N$ values are high throughout the incremental profile, but this may reflect variable adult dietary $\delta^{15}N$ values during this phase which range between 14.7 and 25.5‰ (King et al., 2018b). Az122 T9's $\delta^{15}N$ values fall within the variation seen in other infants and children (younger than 15 years of age) from the Formative Period, which range between 10.7 and 26.6‰ (King et al., 2018a). The $\delta^{15}N$ incremental isotopic profile shape follows the expected changes during weaning, rising after birth, remaining high until around 6 months of age (during the period of exclusive breastfeeding), then decreasing gradually over time as complementary foods are introduced and become an increasingly important part of the diet.

[Figure 4 near here]

Az122 T9 does, however, has several profile features which diverge from the expected form of a weaning curve. Firstly, the value for *in utero* $\delta^{15}N$ (23.0‰) is over five standard deviations higher than the female mean (16.2 ± 1.4‰) for the phase (King et al., 2018b). As Figure 4 shows, we would expect *in utero* $\delta^{15}N$ values to lie close to the phase female mean, although Fuller et al. (2004) show that values may vary up to 1‰ from this due to individual maternal physiology (for example variation in nitrogen retention). These *in utero* values, however, lie beyond this range.

Secondly, the $\delta^{15}N$ profile contains a 5‰ decrease by 3 years of age, followed by a further decrease of almost 2‰ between 3 and 3.5 years of age. A 5‰ decrease in $\delta^{15}N$ is of greater magnitude than the expected 3‰ change, but falls within the range of published values for single trophic level shifts (Caut et al., 2009). The steepness of the further 2‰ decrease at the end of the profile may indicate that processes other than weaning shifts are affecting isotopic values at this age.

Thirdly, the carbon isotopic profile does not echo the changes in the nitrogen isotopic profile. There is an initial slight decrease in $\delta^{13}C$ after birth, followed by rising $\delta^{13}C$ through the period of tooth formation. This contrasts to the expected pattern of an initial rise during breastfeeding then decrease in $\delta^{13}C$ by one trophic level (usually around 1‰) during weaning. In Az122 T9's profile, the steepness of increases in $\delta^{13}C$ is correlated with steepness of decreases in $\delta^{15}N$, i.e. at the end of the profile a sharp decrease in $\delta^{15}N$ is correlated with a sharp increase in $\delta^{13}C$ (Figure 4). Potential interpretations of these unusual aspects of the incremental profile are discussed in detail below.

Discussion

Differential diagnosis:

The new bone formation on the maxilla is non-specific and therefore a differential diagnosis has not been undertaken for this. We note, however, that this kind of bony reaction has been associated with hemorrhage in scurvy (Snoddy et al., 2017). A number of different pathological conditions can cause the porotic hyperostosis and craniosynostosis seen in Az122 T9. These are discussed below and summarized on Table 2.

1. Anemia

Although there is ongoing debate over the specific etiology of porotic hyperostosis (Oxenham & Cavill, 2010; Walker et al., 2009), this lesion has been clinically linked to several forms of anemia (Agarwal et al., 1970; Britton et al., 1960; Resnick, 1995). Anemia is a general term used to describe several disorders of red blood cell dysfunction or underproduction which affect oxygen transport (Resnick, 1995). Anemias can be caused by environmental factors, such as dietary iron or vitamin B12 deficiency, heavy intestinal parasitic load, malaria or other chronic infections, as well as several inherited syndromes such as the thalassemias (Resnick, 1995). In cases of chronic oxygen starvation due to anemia, the body will attempt to compensate by expanding hematopoietic (red blood cell producing) centers of the skeleton, regions rich in trabecular bone (Oxenham & Cavill, 2010; Resnick, 1995). In young children, who exhibit rapid cell turnover, this can lead to porotic hyperostosis of the cranial vault as exhibited by Az122 T9. It is worth noting that the eggs of parasitic organisms known to be associated with B12 deficiency anemia (e.g. *Diphyllobothrium pacificum*) have been found in Chinchorro coprolites from the Arica region (Araújo et al., 2011), which suggests that Az122 T9 may have been at risk for megaloblastic anemia (Walker et al., 2009).

In the nearby Andes high altitude hypoxia during pregnancy has been linked with anemia and porotic hyperostosis lesions (e.g. Rothschild, 2000). However, we consider hypoxia an unlikely cause of the observed porotic hyperostosis. The site of Az122 lies close to sea level. It is possible that Az122 T9 or their mother came from the highlands and experienced hypoxia there, but biodistance studies suggest that there was little movement between the coast and highlands during the Formative Period (Sutter & Mertz, 2004).

Both inherited hemolytic (e.g. thalassemias, sickle cell anemia) and, less commonly, iron deficiency anemia have also been implicated in premature suture fusion during infancy and childhood (Cohen & MacLean, 2000; Duggan et al., 1970). Often all sutures of the skull are involved, resulting in a dome-shaped skull or microcephaly, which usually manifests alongside extensive porotic hyperostosis and/or cribra orbitalia (Duggan et al., 1970). The comorbidity of craniosynostosis with porotic hyperostosis in Az122 T9 is intriguing and these lesions may have a common anemic etiology. As inherited hemolytic anemias do not occur among native South American groups (Ortner, 2003), if there is a common pathological origin for the craniosynostosis and porotic hyperostosis exhibited by this child, it is most likely iron deficiency anemia.

2. Syndromic craniosynostosis

Syndromic craniosynostoses tend to involve more than one suture, as in Az122 T9, and generally have a genetic etiology (Cunningham et al., 2007). Multiple suture synostoses, however, are rarely described in the archaeological literature (see Lewis, 2018). If Az122 T9 has a syndromic form of craniosynostosis it likely also involved changes to the post crania (e.g. changes to the fingers and toes are common in many syndromes – see Table 3). As noted, these elements, are not present for us to use in our differential diagnosis. The expression of craniosynostosis in Az122 T9 does not fit neatly into the diagnostic criteria for any of the major syndromes associated with this condition (Table 3). This is likely because the phenotypic expression of the syndromic craniosynostoses is variable (Morriss-Kay & Wilkie, 2005), with some level of overlap between them. However, Saethre Chotzen Syndrome (Reardon & Winter, 1994) is the condition most consistent with the features observable in Az122 T9.

3. Rickets

Rickets is a general term used to describe a childhood mineralization disorder resulting from vitamin D, calcium, and/or phosphorus deficiency, as well as several inborn disorders of mineral metabolism (Ortner, 2003). Vitamin D deficiency rickets may result in craniosynostosis, the expression of which is linked to the severity of the micronutrient deficiency in the individual (Shashi & Hart, 2002). Az122 T9 is missing post-cranial elements which exhibit the most strongly diagnostic lesions of rickets (Brickley & Ives, 2008). However, vitamin D deficiency is unlikely given the UV-B-rich environment of the Atacama Desert. As such, although we cannot exclude the possibility of rickets in this individual, we argue that other causes of craniosynostosis are more likely in this case.

4. Hyperthyroidism

Hyperthyroidism can result in premature fusion of the sagittal and lambdoid sutures, and partial fusion of the coronal suture (Segni et al., 1999). This pattern of sutural involvement is similar to that of Az122 T9, although in this individual the entire coronal suture is obliterated rather than just the upper third expected with hyperthyroidism (Shashi & Hart, 2002). We also consider this an unlikely etiology as hyperthyroidism is extremely rare in children under 5 years of age (Segni et al., 1999).

5. Artificial cranial deformation

Craniosynostosis has previously been reported in archaeological populations of the Americas associated with the cultural practice of artificial cranial deformation (O'Loughlin, 1996; White, 1996). Cranial constraint can cause premature sutural fusion, or increased complexity of sutures (O'Loughlin, 2004), but is also associated with quite severely asymmetric cranial shapes (reflecting the direction of imposed stress) (White, 1996). The cranial shape of Az122 T9 does not appear to have been artificially modified. Although it is small, the asymmetry is

not unidirectional. We therefore consider artificial cranial deformation one of the least likely causes of craniosynostosis in this child.

6. Cyanotic congenital heart disease

Cyanotic congenital heart disease (CCHD) is a clinically rare condition, occurring in 0.15% of live births (Renno & Johns, 2018). CCHD is associated with a number of cardiac lesions that result in obstruction of blood to the pulmonary arteries (Renno and Johns, 2018). Porotic hyperostosis has been clinically observed in untreated cases of CCHD and, like the anemias, is thought to occur as the body's osseous response to chronic oxygen starvation (Walor et al., 2005). Intriguingly, premature fusion of the sagittal and coronal sutures has also been reported in a case study of CCHD in an eight-year-old child (Walor et al., 2005). Given the extreme clinical rarity of this condition we argue that it is an unlikely cause of the lesions exhibited by Az122 T9.

Possible evidence for in utero stress:

In Az122 T9 the *in utero* forming dentine increment had a $\delta^{15}N$ value over 2‰ and 5SD above the female mean for the phase. It is possible that this reflects increased maternal protein intake at the end of pregnancy, or a mother with a high reliance on marine foods in general causing elevated $\delta^{15}N$ (King et al., 2018a). Alternatively, multiple studies have shown that $\delta^{15}N$ may also be raised during periods of maternal stress or catabolism of maternal tissues to meet the energy needs of the unborn child. Fuller et al. (2005), for instance noted increased maternal $\delta^{15}N$ values during periods of morning sickness, with a return to normal dietary values as gestation progressed. However, mothers experiencing physiological stress during their last trimester retained high values, resulting in high *in utero* $\delta^{15}N$ values. Similarly raised $\delta^{15}N$ has been noted in individuals suffering from nutritional deprivation (Mekota et al., 2006), and has been interpreted as evidence for stress in historic famine populations (Beaumont & Montgomery, 2016; Beaumont et al., 2015).

It is possible that, if the craniosynostosis present in Az122 T9 is of genetic origin (i.e. syndromic or hereditary condition such as hemolytic anemia), this would have begun manifesting *in utero* (Delahaye et al., 2003; Urbaniak & Greiss, 2000). The premature fusion of cranial sutures may therefore have acted as a stressor, contributing to a physiological response during this period.

If Az122 T9 was exposed to stress *in utero* it is likely to have had implications for their post-natal development, particularly if that stress involved either general undernutrition or a specific nutritional deficiency (Barker, 1998; Mulder et al., 2002; Schneider et al., 1999). In the case of Az122 T9, for whom iron-deficiency anemia is a plausible issue, it is possible that maternal nutrient deficiency led to low fetal iron stores which would have been further depleted during infancy (Allen, 2000; Colomer et al., 1990).

Evidence for stress throughout infancy and childhood:

All of the incremental isotopic values for Az122 T9 fall well above the adult female dietary values for the phase, even with the decrease in values (from around 24 to 17‰), which we interpret as representing weaning. This could be considered evidence that stress throughout infancy and childhood is raising isotopic values. Adult male values within the Formative Period, however, range up to 25.5‰, higher than any of the values in Az122 T9's tissues. It is, therefore, possible that high values in this individual are purely due to maternal diet being rich in high $\delta^{15}N$ resources such as marine foods, and weaning onto a diet that reflects these adult dietary inputs.

The extensive pathology exhibited by this individual, however, indicates that pathological processes are likely to have been ongoing for much of the individual's life. Although children typically manifest lesions more rapidly than adults (Lewis, 2018), the osseous changes observed in Az122 T9 are suggestive of a chronic state of physiological stress, lasting months or years rather than days or weeks. We therefore consider it likely that generally high $\delta^{15}N$ reflects a combination of a high $\delta^{15}N$ dietary baseline, and chronic stress experienced throughout infancy and early childhood.

It is possible that the slight decrease in $\delta^{13}C$ just after birth may also be evidence for nutritional stress in the mother or infant. Recent research suggests that $\delta^{13}C$ does not necessarily track the weaning process (Herrscher et al., 2017), and may instead be more affected by nutritional stress. For instance, the breakdown of fat deposits with low $\delta^{13}C$ to meet energy requirements may cause decreases in $\delta^{13}C$ (Mekota et al., 2006). However, factors causing minor changes to $\delta^{13}C$ during breastfeeding are poorly understood. We therefore cannot be certain that the small change in $\delta^{13}C$ values just after Az122 T9's birth is stress related.

Evidence for a micronutrient deficient weaning diet and its links to the visible pathology:

The incremental isotopic profile of Az122 T9 is also unusual in that its $\delta^{13}C$ values rise as $\delta^{15}N$ decreases, rather than also decreasing by the expected 1‰ during weaning. The most parsimonious explanation for this is that the child was weaned onto C_4 resources, which did not form such a significant component of the maternal diet (Dupras et al., 2001; Dupras & Tocheri, 2007; Katzenberg et al., 1993; Sandberg et al., 2014; Wright & Schwarcz, 1998), thus causing their values to rise in relation to the composition of breastmilk. In the Atacama context maize is the major C_4-crop, and there are no endemic C_4 resources. Maize gruel is also well-documented as a weaning food worldwide (Gibson et al., 1998; Onofiok & Nnanyelugo, 1998), with isotopic evidence for its use throughout the Americas (Buikstra et al., 1986; Finucane, 2009).

Maize, while being an easily digestible, readily available source of supplementary nutrition, is protein deficient and has a low iron bioavailabilty (Layrisse et al., 1990; Osborne & Mendel, 1914). This low availability of iron in complementary foods is responsible for high rates of iron-deficiency anemia in infants and young children in developing countries today (Dewey, 2007; Zimmermann & Hurrell, 2007), and likely had similar effects in the past. Although

preparation methods (such as alkali processing or soaking of grains) can be used to increase iron uptake from maize, there is no ethnographic evidence for the use of these techniques in Andean or Atacama populations (Katz et al., 1974). The broad-spectrum adult diet would have provided iron from other resources. However, infants and children, like Az122 T9, who were weaned onto a single cereal crop are likely to have been vulnerable to iron-deficiency anemia.

There is paleopathological evidence suggestive of iron deficiency in Az122 T9. The carbon isotope profile from dentinal collagen gives a possible mechanism for this deficiency, and suggests that not only was the individual weaned using C_4 complementary foods, but that the diet of the individual became even more C_4-based after the age of three, indicated by the further steep increase in $\delta^{13}C$ and decrease in $\delta^{15}N$ values at this point in time. Indeed, the fact that the porotic hyperostosis lesions were still active at time of death (5 years) suggests that this deficient diet continued even up to the time of death.

Summary:
The interactions between infant-feeding practices and pathology mean that pathological individuals are often not studied isotopically. However, the incremental isotopic profile of Az122 T9 highlights the potential for infant-feeding practices to be visible isotopically despite pathology. The isotopic profile echoes the expected changes in $\delta^{15}N$ values for 'normal' weaning, beginning at 6 months of age, with the weaning process lasting until at least 3 years of age. There is also isotopic evidence for stress prior to the weaning process in the form of high *in utero* $\delta^{15}N$, and evidence that complementary feeding choices resulted in micronutrient deficiencies in the individual. Maize gruel supplementation is a possible causative factor for iron-deficiency anemia, the bony evidence for which exists in the form of porotic hyperostosis.

Az122 T9 is also interesting from a bioarchaeology of care perspective (Tilley & Schrenck, 2017), as an individual who survived for a length of time while experiencing quite severe health issues. Developing an index of care for Az122 T9 is outside of the scope of this project, but is an avenue for future research. This approach considers the clinical implications of bioarchaeologically observed skeletal pathology, and understanding of the level of care needed for the individual's survival, and social implications of this care. In this case initial examination of the clinical literature suggests that many of the syndromic synostoses (including Saethre Chotzen Syndrome) can be associated with abnormal development of the intestinal tract (Hibberd et al., 2016). Similarly, cyanotic congenital heart disease can be associated with feeding difficulties in early life (Jadcherla et al., 2009). Development of this research may better reveal potential links between the observed pathologies and the nutritional deficiencies inferred from isotopic analyses in Az122 T9.

Conclusion

Both infant-feeding practices and the experience of periods of physiological stress due to other factors in childhood have significant implications for adult health and population dynamics. Until recently, however, it has been difficult to definitively associate weaning behaviors with evidence of stress in the skeleton. In this study, we show that incremental isotopic analysis has

the potential to show *in utero* stress transfer, when weaning begins and how long the process extends for, complementary feeding practices, and childhood diet. We examine the isotopic evidence for weaning in combination with paleopathological indicators for health, showing that a micronutrient deficient weaning diet likely had implications for childhood health. This case study highlights the uses of both combining multiple lines of evidence to build a full picture of the synergies between diet and health, and recognizing individual experience in the past.

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Table 1: Carbon and nitrogen stable isotope ratio data and quality control parameters for increments from Az122 T9, maxillary left dM_1. The mid-point ages represented by each increment were calculated using Beaumont and Montgomery's (2015) method.

Increment	Age represented by increment (years)	$\delta^{13}C$ (‰ PDB)	$\delta^{15}N$ (‰ AIR)	%C	%N	C:N
1	-0.2	-15.8	23.0	41.2	14.5	3.3
2	0.2	-16.1	23.7	42.4	14.6	3.4
3	0.6	-15.9	23.8	42.3	15.0	3.3
4	1.0	-15.6	23.3	41.7	14.8	3.3
5	1.4	-15.6	22.5	42.3	15.0	3.3
6	1.9	-15.5	21.7	42.1	14.9	3.3
7	2.3	-15.0	21.0	41.9	14.9	3.3
8	2.7	-14.8	20.5	41.7	14.7	3.3
9	3.1	-14.5	19.6	39.3	14.9	3.1
10	3.5	-13.9	17.3	42.7	14.6	3.4

Table 2: Summary of potential pathological and cultural conditions which could result in the porotic hyperostosis and craniosynostosis observed in Az122 T9.

Condition	Skeletal manifestation	Etiology	Source
Anemia	Porotic hyperostosis Cribra orbitalia Rarely premature fusion of cranial sutures	Dietary iron or Vit B12 deficiency High intestinal parasite load	(Duggan et al., 1970; Oxenham & Cavill, 2010; Resnick, 1995)

		Malaria or other severe infections. Inherited disorder (e.g. thalassemia).	
Rickets	Low bone mineral density (osteopenia). Endochrondral growth defects. Porous ectocranial new bone (healing rickets).	Vit D, Ca or P deficiencies. Inborn errors of Vit D metabolism Renal, hepatic, or intestinal disease.	(Brickley & Ives, 2008)
Hyperthyroidism	Few clear skeletal effects, but can involve premature cranial suture fusion.	Maternal thyroid disorders. Selenium depleted diet.	(Segni et al., 1999; Shashi & Hart, 2002)
Syndromic craniosynostoses	See Table 3. All involve premature fusion of cranial sutures	Generally genetic etiology	See Table 3
Cranial deformation	Altered cranial morphology. Premature suture fusion Increased complexity of sutures.	Cultural modification (binding etc.) .	(O'Loughlin, 2004; White, 1996)
Cyanotic Congenital Heart Disease	Possibly porotic hyperostosis and/or craniosynostosis.	Genetic etiologies.	(Renno & Johns, 2018; Walor et al., 2005)

Table 3: Syndromic etiologies of craniosynostosis presented with reference to Az122 T9. Diagnostic information compiled from clinical studies (Cohen & MacLean, 2000; Delahaye et al., 2003; Dodge et al., 1959; Jabs, 2002; Reardon & Winter, 1994).

Cause of Craniosynostosis	**Features**	**Present (✓), absent (×) or unobservable (–) in Az122 T9**	**Comments**
Crouzon Syndrome	Craniosynostosis (usually coronal and sagittal). Dome-shaped skull.	✓	

	Underdeveloped midface, involving receding maxilla. Ocular proptosis, caused by shallow orbits	✓ ✗	
Apert Sydrome	Craniosynostosis (coronal, sagittal, lambdoid). Underdeveloped midface. Prominent forehead (dome-shaped skull). Shallow orbits. Symmetric syndactyly of the hands and feet.	✓ ✓ ✓ ✗ –	
Pfieffer Syndrome	Craniosynostosis (coronal and sagittal). Underdeveloped midface. Shallowed orbits. Elbow synostosis. Broad thumbs and big toes and variable syndactyly.	✓ ✓ ✗ – –	More sutures involved than is typical for this syndrome
Jackson-Weiss Syndrome	Craniosynostosis (coronal and sagittal). Midface underdevlopement. Abnormalities of the feet (key diagnostic feature).	✓ ✓ –	Craniosynostosis probably more severe than would be expected in this syndrome
Muenke Syndrome	Craniosynostosis (usually only uni/bilateral coronal suture). Midface underdevelopment. Brachdactyly and carpal/tarsal coalition.	✓ ✓ –	More sutures involved than is typical for this syndrome
Saethre Chotzen Syndrome	Craniosynostosis (most commonly coronal suture, but any/all can be involved) resulting in brachycephaly or dome-shaped skull. Cranial Assymetry. Deviated nasal septum. Slight underdeveloped midface. Brachydactyly.	✓ ✓ ✓? ✓ –	Deviated nasal septum not really observable, but assymetry of nasal region suggests it was present

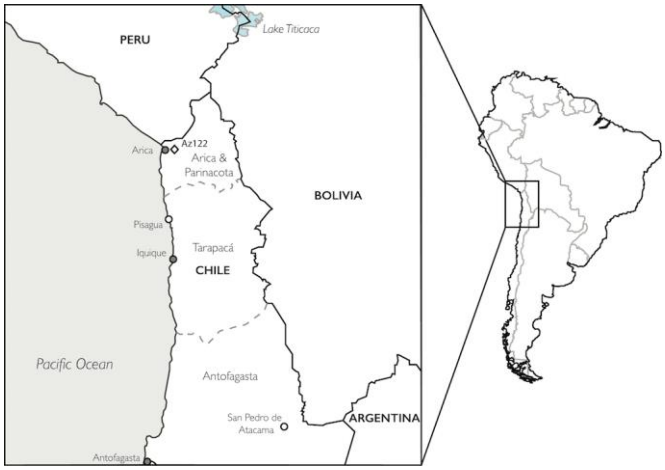

Figure 1: The location of Az122 relative to current geographic borders and modern cities. Adapted from King et al. (2018b).

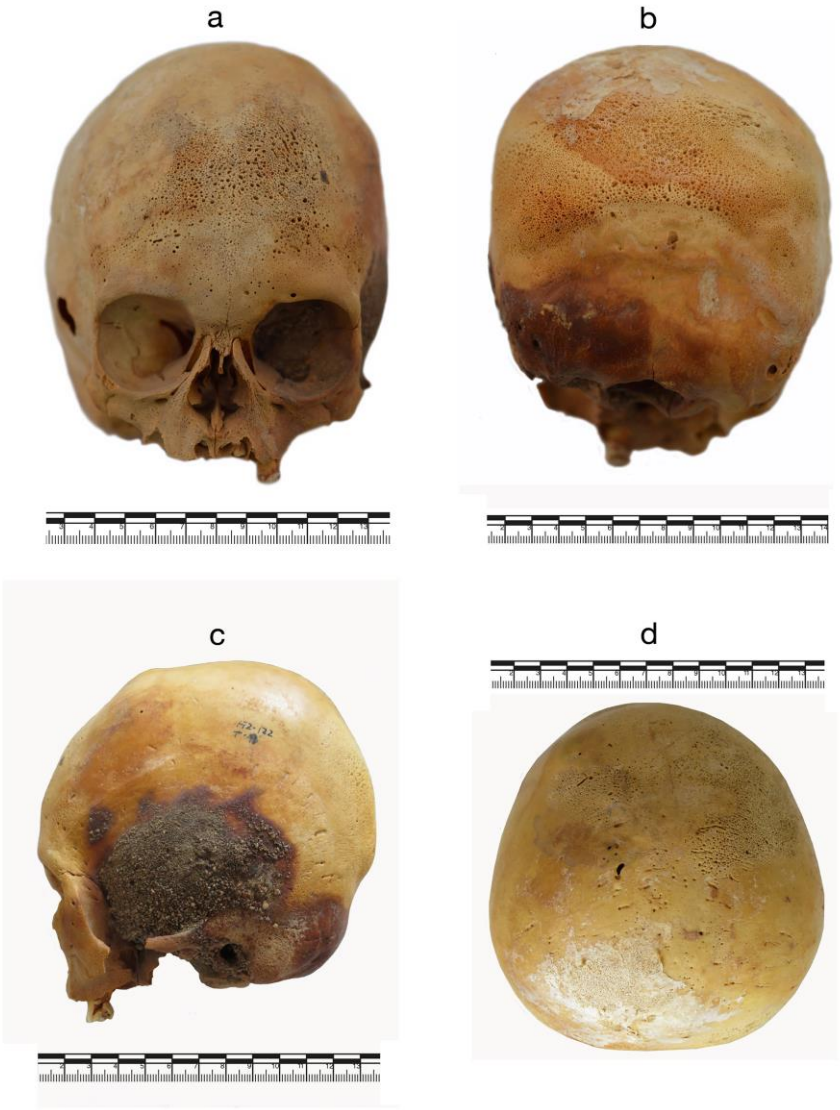

Figure 2: Anterior (a), posterior (b), left lateral (c) and superior (d) aspects of the cranium of Az122 T9, showing extensive porotic hyperostosis, asymmetry, dome-shaped skull and obliteration of cranial sutures.

Figure 3: Lateral view radiograph of the cranium of Az122 T9. The image shows "hair-on-end" hypertrophy (accentuated vertical trabeculae between the inner and outer tables) in the occipital and frontal bones (arrows). The trabeculae (diplöe) is visibly expanded in the occipital. The endocranium also shows several depressions and thinning of the tables, likely relating to premature suture fusion and intracranial pressure.

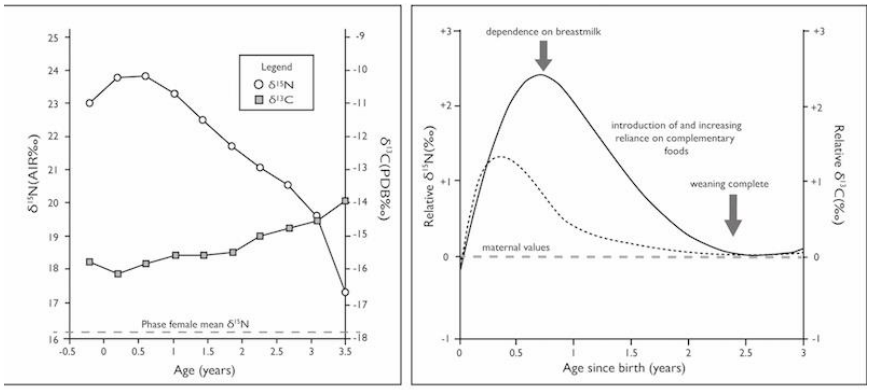

Figure 4: Incremental isotopic profile of Az122 T9 (left), compared with the ‘expected’ weaning curve for an individual weaning onto a diet similar to that of their mother (right; adapted from Halcrow et al. (2018) and Jay et al. (2008)). Note that while the shapes of the $\delta^{15}N$ curves look similar, the y-axis is scaled differently because the decrease in $\delta^{15}N$ in Az122 T9 is larger than expected.